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Obstruction of endotracheal tube with relevant respiratory acidosis during pediatric cardiac surgery

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Key points

Nasal intubation for cardiac surgery in children requiring systemic anticoagulation can lead to obstruction of the endotracheal tube by clot formation, requiring immediate tube replacement.

Abstract

We describe a case of pediatric cardiac surgery in a 21-days old baby, in whom a nasal endotracheal tube (ETT) was inserted. At the end of surgery both ventilatory pressures and end-tidal CO_2 increased suggesting airway obstruction. Suctioning of the ETT lumen did not relieve the problem, only ETT replacement did. The ETT was almost completely obstructed with a clot, leading to significant respiratory acidosis. We would like to bring awareness of the possibility of ETT obstruction in pediatric cardiac surgery with nasal intubation and systemic anticoagulation, in which only tube exchange relieved the problem.

Keywords: airway obstruction; pediatric cardiac surgery, nasal intubation, systemic anticoagulation

Background

In congenital pediatric cardiac surgery, especially in neonates, the patient is often intubated nasally in order to prevent endotracheal tube malposition and accidental spontaneous extubation in the intensive care unit. However, nasal intubation together with the need for systemic anticoagulation with heparin for cardiac surgery is a known risk factor for endotracheal tube obstruction.

Patient case description

A 21-days old baby, length 50 cm, weight 3 kg, with the diagnosis of total anomalous pulmonary venous return (TAPVR) was scheduled for TAPVR repair. He was cyanotic, had respiratory distress and rapid breathing despite supplementary oxygen breathing. The echocardiography showed next to the TAPVR, in which all four pulmonary veins drained abnormally to the right atrium instead of the left atrium, dilatation and hypertrophy of the right ventricle, an open foramen ovale with continuous right to left shunt and a relatively small left atrium and left ventricle, and a patent ductus arteriosus. On the day of surgery anesthesia was induced with sevoflurane through a breathing mask and intravenous injection of sufentanil 2 microgram per kg, midazolam 0.3 microgram per kg and rocuronium 1.2 mg per kg followed

by a nasal intubation with a cuffed endotracheal tube (ETT) sized 3 mm (internal diameter), distance 11.5 cm from the nostril. The intubation was easy and succeeded in one attempt. Auscultation of the lungs revealed a good position of the ETT with symmetric bilateral vesicular sounds. Anesthesia was maintained with midazolam and sufentanil. An arterial line was inserted in the right femoral artery and a central venous line in the right internal jugular vein. The peak inspiratory pressure was 19 cmH_2O, end-tidal carbon dioxide ($ETCO_2$) was 5.0 kPa. Blood gas analysis showed a pH of 7.41, a PaO_2 of 7.6 kPa, a $PaCO_2$ of 6.1 kPa and an arterial oxygen saturation of 92%. Sternotomy was performed and after opening of the pericardium the patient was placed on cardiopulmonary bypass using arterial cannulation of the ascending aorta and venous cannulation of both the superior and inferior vena cava. Patient was heparinized (4 mg/kg) and surgery was performed during cardiopulmonary bypass for 3.5 hours and consisted of correction of total anomalous pulmonary venous connection, according to the Tucker technique, ligation of the patent ductus arteriosus, ligation of the vertical vein, and patch-closure of the atrial septal defect using autologous pericardial patch. During this procedure a period of 25 minutes of circulatory arrest was needed to reach the collector of the pulmonary veins located posterior to the pericardium. The patient was cooled to a oesophageal temperature of 23° Celsius. After complete re-warming of the patient, weaning from cardiopulmonary bypass was uneventful. The systemic arterial blood pressure was 65/35 mmHg while the pulmonary arterial pressure was between 30-35 mmHg. The left atrial pressure was between 8 and 12 mmHg. An epicardial echocardiogram was performed, showing good size of the left atrial chamber, and a good biventricular function. The anastomotic side was identified and found to be free from obstruction. Protaminsulfate was then administered (15 mg) and decannulation was achieved. During the hemostasis period the peak inspiratory pressure increased gradually from 22 to 44 cmH_2O on the ventilator, the $ETCO_2$ varied between 4 and 7 kPa. After exclusion of kinking and malposition of the endotracheal tube, suctioning of the ETT for the presence of secretion or blood was performed but was negative. On auscultation of the lungs a vesicular breathing sound was heard with wheezes on both sides. Manual ventilation of the lungs revealed some resistance. An allergic reaction to protamine was briefly considered but eventually not suspected because of the good hemodynamic picture. The blood gas analysis showed a gradual increase of $PaCO_2$ from 6.1 to 17.6 kPa with a decline of pH from 7.31 to 6.92. Thus, the patient experienced a period of respiratory acidosis through retention of CO_2. The PaO_2 and arterial oxygen saturation (SaO_2) remained adequate (30.4 kPa and 99%, respectively). Cerebral oxygenation (rSO_2) as obtained by near-infrared spectroscopy (Invos 5100C, Covidien, Dublin, Ireland) nicely followed the changes in SaO_2 but never decreased below 50%. After all failed attempts to reduce the $PaCO_2$ the endotracheal tube was exchanged and the tip of the ETT was found to be almost completely obstructed with a big clot (Fig. 1).

Fig. 1. Photography of the removed endotracheal tube, which is almost completely obstructed with a clot.

After placement of a new endotracheal tube, ventilatory pressures and $PaCO_2$ decreased immediately while systemic (SaO_2) and regional oxygenation (rSO_2) increased to almost normal values.

Discussion and conclusions

This case report shows the relevant metabolic consequences of a partly obstructed ETT, which is of utmost importance especially with small tubes used in children.

Of note, suction of the ETT failed to relief the obstruction. The patient had an enormous drop in the pH to a minimum of 6.92. This drop could have led to a cardiac arrest if not identified and corrected in time. In addition, the huge increase in $PaCO_2$ did not correspond with an equivalent increase in end-tidal CO_2. While an increase in peak airway pressure is a typical warning sign for (partial) ETT obstruction,[1] the differential diagnosis for high peak airway pressures and CO_2 retention includes kinking, malposition and migration of the ETT, obstruction of the tube filter with blood or secretion, presence of long edema, atelectasis, pleural effusion, bronchospasm, pneumothorax and an allergic reaction to drug administration.

In this case it was hazardous to us that suctioning of the ETT did not show any blood or secretion, perhaps because of the firm clot formation and the small diameter of the tube.

In the literature there are few case reports over obstruction in the ETT by blood clot, mucus and even by overinflation of the cuff.[2-5]

Nasal intubation can cause damage to the mucosa of the nasal cavity and turbinate. We conclude that nasal intubation for cardiac surgery with full heparinization is a risk factor for bleeding and ETT occlusion with clot, which can lead to relevant respiratory acidosis

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References

1. Kawati R, Lattuada M, Sjostrand U et al. Peak airway pressure increase is a late warning sign of partial endotracheal tube obstruction whereas change in expiratory flow is an early warning sign. Anesth Analg 2005; 100: 889-893

2. Arney KL, Judson MA, Sahn SA. Airway obstruction arising from blood clot: three reports and a review of the literature. Chest 1999; 115: 293-300

3. Xue FS, Luo MP, Liao X et al. Delayed endotracheal tube obstruction by mucus plug in a child. Chinese Medical Journal 2009; 122: 870-872

4. Johnson KM, Lehman RE. Acute management of the obstructed endotracheal tube. Respir Care 2012; 57: 1342-1344

5. Lim HK, Lee MH, Shim HY et al. Complete obstruction of an endotracheal tube due to an unexpected blood clot in a patient with a hemopneumothorax after repositioning of the patient for lumbar spine surgery. Korean Journal of Anesthesiology 2013; 64: 382-383